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Introduction

Cholesterol and triglycerides are important fats (lipids) in the blood. Cholesterol is an essential component of cell membranes, brain and nerve cells, and bile, which helps the body absorb fats and fat-soluble vitamins. The body uses cholesterol to make vitamin D and various hormones, such as estrogen, testosterone, and cortisol. The body can produce all the cholesterol that it needs, but it also obtains cholesterol from food. Triglycerides, which are contained in fat cells, can be broken down, then used to provide energy for the body's metabolic processes, including growth. Triglycerides are produced in the intestine and liver from smaller fats called fatty acids. Some types of fatty acids are made by the body, but others must be obtained from food (see Overview of Nutrition: Fats).

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Pronunciations

atherosclerosis

chylomicrons

hypolipoproteinemia

lipoproteins

pancreatitis

Fats, such as cholesterol and triglycerides, cannot circulate freely in the blood, because blood is mostly water. To be able to circulate in blood, cholesterol and triglycerides are packaged with proteins and other substances to form particles called lipoproteins.

There are different types of lipoproteins. Each type has a different purpose and is broken down and excreted in a slightly different way. Lipoproteins include chylomicrons, very low density lipoproteins (VLDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL). Cholesterol transported by LDL is called LDL cholesterol, and cholesterol transported by HDL is called HDL cholesterol.

The body can regulate lipoprotein levels (and therefore lipid levels) by increasing or decreasing the production rate of lipoproteins. The body can also regulate how quickly lipoproteins enter and are removed from the bloodstream.

Levels of cholesterol and triglycerides vary considerably from day to day. From one measurement to the next, cholesterol levels can vary by about 10%, and triglyceride levels can vary by up to 25%.

Lipid levels may become abnormal because of changes that occur with aging, various disorders (including some hereditary ones), use of certain drugs, or lifestyle (consuming a high-fat diet, being physically inactive, or being overweight).

Abnormal levels of lipids (especially cholesterol) can lead to long-term problems, such

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as atherosclerosis. Generally, a high total cholesterol level (which includes LDL, HDL, and VLDL cholesterol) or a high level of LDL (the "bad") cholesterol increases the risk of atherosclerosis and thus the risk of heart attack and stroke. However, not all types of cholesterol increase this risk. A high level of HDL (the "good") cholesterol may decrease risk, and conversely, a low level of HDL cholesterol increases risk. The effect of triglyceride levels on the risk of heart attack is less clear-cut. But very high levels of triglycerides (higher than 500 milligrams per deciliter of blood, or mg/dL) can increase the risk of pancreatitis. For people older than 20, levels of total cholesterol, triglycerides, LDL cholesterol, and HDL cholesterol after fasting should be measured at least once every 5 years. Collectively, these measurements are called the fasting lipoprotein profile.

Lipoproteins: Lipid Carriers

Type	Formation	Lipid Content	Function
Chylomicrons	Formed from fats in food processed by the intestine	Mostly triglycerides	To transport digested fats (as triglycerides) to muscle and fat cells
Very low density lipoprotein	Formed in the liver	More than $^1/_2$ triglycerides About $^1/_4$ cholesterol	To transport triglycerides from the liver to fat cells
Low-density lipoprotein	Formed from VLDL after it delivers triglycerides to fat cells	More than $^1/_2$ cholesterol Less than $^1/_{10}$ triglycerides	To transport cholesterol to various cells
High-density lipoprotein	Formed in the liver and small intestine	About $^1/_4$ cholesterol About $^1/_{20}$ triglycerides	To remove cholesterol from tissues in the body and transport it to the liver

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Cerebral blood flow velocity and systemic vascular resistance after acute reduction of low-density lipoprotein in familial hypercholesterolemia.

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BACKGROUND AND PURPOSE: Low-density lipoprotein apheresis is currently used for the treatment of familial hypercholesterolemia, an inherited disorder of metabolism associated with premature development of cardiovascular disease. We wanted to evaluate cerebral blood flow velocity, cardiac output, and systemic vascular resistance in patients with familial hypercholesterolemia before and after low-density lipoprotein apheresis. METHODS: Ten patients (age range, 14 to 46 years; 4 males, 6 females) with familial hypercholesterolemia (8 homozygotes, 2 heterozygotes) and 10 healthy control subjects of comparable age and sex distribution participated in the study. Low-density lipoprotein apheresis by dextran sulfate was performed in 8 patients (7 homozygotes, 1 heterozygote). Six patients (4 homozygotes, 2 heterozygotes) underwent a procedure of extracorporeal erythrocyte filtration with the same extracorporeal volume as for low-density lipoprotein apheresis, but with the exclusion of the passage of plasma through the dextran sulfate column. Cerebral blood flow velocity (transcranial Doppler), cardiac output, and systemic vascular resistance (electric bioimpedance cardiography) were determined by noninvasive techniques before and 1 day and 7 days after low-density lipoprotein apheresis or extracorporeal erythrocyte filtration. Plasma and blood viscosities were measured at the same time. RESULTS: Before apheresis, mean and diastolic cerebral flow velocities were abnormally low in hypercholesterolemic patients ($P < .01$ and $P < .02$ vs healthy control subjects, respectively). After apheresis, low-density lipoprotein cholesterol was lowered by 40% to 60% from baseline, and cerebral blood flow velocities (mean, systolic, and diastolic velocities) were increased ($P < .01$). Cardiac output, systemic vascular resistance, and viscosity values were not significantly modified. Extracorporeal erythrocyte filtration (without passage of plasma through the dextran sulfate column) did not modify serum lipids, hemodynamic parameters, or viscosity values. CONCLUSIONS: Low-density lipoprotein apheresis produces potentially useful hemodynamic effects. They are not adequately explained by changes in blood viscosity alone and might reflect a restoration of endothelium-mediated vasodilation, which is inhibited by high concentrations of low-

density lipoprotein.

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